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The evaluation of microbiology and Fournier's gangrene severity index in 27 patients

Mehmet Uluğ [b], **Ercan Gedik** [a,*], **Sadullah Girgin** [a], **Mustafa K. Çelen** [b], **Celal Ayaz** [b]

[a] *Department of General Surgery, Dicle University Medical School, 21280 Diyarbakır, Turkey*
[b] *Department of Infectious Diseases and Clinic Microbiology, Dicle University Medical School, 21280 Diyarbakır, Turkey*

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Summary
Objectives: The objectives of this study were to identify the causative microorganisms and factors associated with survival in patients with Fournier's gangrene and to determine the accuracy of the Fournier's gangrene severity index.
Materials and methods: We retrospectively evaluated 27 patients with Fournier's gangrene who were treated and followed up at our hospital between January 2005 and December 2006. Biochemical, hematologic, and bacteriologic study results at admission and at the final evaluation, etiologic and predisposing factors at admission, physical examination findings, the timing and extent of surgical debridement, and antibiotic therapy used were all recorded.
Results: The admission laboratory parameters that were significantly correlated with outcome included urea, creatinine, sodium, and potassium; at the final evaluation, in addition to these parameters, hematocrit, albumin, and bicarbonate levels were also significantly associated with outcome. The mean Fournier's gangrene severity index score (FGSIS) at admission for survivors was 5.04 ± 2.49 compared with 13.6 ± 4.61 for non-survivors. There was a strong correlation between the FGSIS and mortality ($p < 0.0001$). *Escherichia coli* and *Pseudomonas aeruginosa* were the most commonly isolated microorganisms.
Conclusions: Patient metabolic status and predisposing factors are important in the prognosis of Fournier's gangrene. Hence, we believe that the FGSIS should be used clinically to evaluate therapeutic options and assess results.

Introduction

Fournier's gangrene (FG) is a life-threatening necrotizing fasciitis of the perineal, genital and perianal region, which leads to thrombosis of the small subcutaneous vessels and

* Corresponding author.
E-mail address: egedik@dicle.edu.tr (E. Gedik).

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results in the development of gangrene of the overlying skin.[1] FG was first described in 1883 by Jean Alfred Fournier, who was a French venereologist. Fournier concluded that three findings characterize the syndrome: abrupt onset in a healthy young man, rapid progression, and the absence of a specific causative agent.[2,3] In cases originating in the genitalia, the infecting bacteria probably pass through Buck's fascia of the penis and spread along the Dartos fascia of the scrotum and penis, Colles' fascia of the perineum, and Scarpa's fascia of the anterior abdominal wall.[4]

Despite advancements in knowledge regarding the etiology, diagnosis, modern intensive care techniques, and medical therapy of FG, the mortality rate remains high at between 0% and 67%.[2,5,6] Although conservative treatment without surgery may suffice in some cases, FG remains a surgical emergency, and early recognition and prompt radical debridement are the mainstays of management.

It is believed that systemic conditions such as age, diabetes mellitus, alcoholism, paralysis or neurological deficit, malignancy, debility, and immunosuppression predispose to the development of this entity.[7,8]

In published reports, the factors affecting survival or death from FG were not clearly identified until the study of Laor et al. in 1995, in which measurable pathophysiologic data between survivors and non-survivors of FG were compared.[9]

We reviewed our experience of 27 patients treated for FG to identify the factors affecting survival and the microbiology of the disease.

Materials and methods

The medical records of 27 consecutive patients with FG, who were treated and followed up at the departments of General Surgery and Infectious Diseases and Clinic Microbiology, Dicle University Hospital, Diyarbakır, Turkey, between January 2005 and December 2006, were reviewed retrospectively.

Clinical diagnoses were based on general appearance, medical history, physical examination, laboratory data, and signs and symptoms upon admission. Biochemical, hematologic, and bacteriologic study results at admission and at the final evaluation, etiologic and predisposing factors at admission, physical examination findings, the timing and extent of surgical debridement, and antibiotic therapy used were all recorded. The admission time was defined as the time interval from the onset of symptoms to hospital admission.

The extent of gangrene was calculated for a modified body surface area in nomograms routinely used to assess the extent of burn injuries: the penis, scrotum, and perineum accounted for 1% surface area, the ischiorectal fossa for 2.5%, etc.

All culture samples were transported without delay to our clinical laboratory. The material was routinely streaked onto eosin–methylene blue agar and 5% sheep blood agar. The two plates were incubated in air at 37 °C for 24 hours for aerobic microorganisms. Further eosin–methylene blue agar and 5% sheep blood agar plates were sealed into Ziploc plastic bags to maintain an increased CO_2 atmosphere at 37 °C for 24–72 hours for anaerobic microorganisms. Before the results were obtained, a wide range of antibiotherapy was started empirically in combinations of penicillin G, third-generation cephalosporin, especially ceftriaxone, and metronidazole intravenously, according to the general condition of the patient. These regimens were changed on the basis of sensitivity tests.

All patients underwent early, aggressive surgical debridement, and additional debridements were performed when necessary. For those who survived, no additional procedures were needed, and the wounds were followed up for secondary healing.

The Fournier's gangrene severity index (FGSI), which was created by modifying the acute physiology and chronic health evaluation II severity score (APACHE II) by Laor et al. in 1995, was used in our study.[9] The index was developed in an attempt to assign a numerical score that describes the severity of the FG. In the FGSI, nine parameters are measured, and the degree of deviation from normal is graded from 0 to 4. The individual values are summed to reach the FGSI score (FGSIS). These parameters are temperature, heart rate, respiratory rate, serum sodium, potassium, creatinine, and bicarbonate levels, hematocrit, and leukocyte count (Table 1). The data were assessed according to whether the patient survived or not.

Statistical analysis was performed with SPSS for Windows, version 11.0 (SPSS Inc., Chicago, IL, USA). All data were analyzed by the Mann–Whitney U-test. The admission and final evaluation parameters for each group were compared by Wilcoxon signed-rank test. *p*-Values of less than 0.05 were considered statistically significant.

Results

The mean age of patients who survived was 53.95 ± 21.49 (range 19–88) years and for non-survivors was 57.20 ± 12.94 (range 38–73) years (Figure 1 and Table 2). The difference in age between survivors and non-survivors was not significant ($p = 0.050$, $U = 24\,000$). Of the patients, 20 (74.1%) were men and seven (25.9%) were women, and two of the non-survivors were women. Of the 27 patients studied, 22 survived and five died; the overall mortality rate was 18.5%.

Presenting symptoms included erythema in 27 patients (100%), perianal/scrotal swelling in 23 patients (85.1%), perianal/scrotal pain in 21 patients (77.8%), purulence or wound discharge in 19 patients (70.4%), and fever in 12 patients (44.4%).

The median extent of the body surface area involved in the necrotizing process in patients who survived was 2.29% and in patients who did not survive was 3.4% ($p = 0.084$, $U = 28\,000$). The difference in median admission time was not significant between survivors and non-survivors; it was 6.68 ± 3.73 (range 2–15) days for survivors and 5.4 ± 3.43 (range 2–10) days for non-survivors ($p = 0.395$, $U = 41\,500$). All patients underwent radical surgical debridement with excision of all necrotic material and tissue of doubtful viability. The number of surgical debridements did not seem to influence patient outcome; it was performed a mean of 1.59 times in patients who survived compared to 1.4 times in patients who subsequently died. This difference was not statistically significant ($p = 0.599$, $U = 47\,500$).

The laboratory parameters measured at admission and at final evaluation are given in Table 2, and included the white blood cell count, hematocrit, platelet count, and serum urea, creatinine, sodium, potassium, alkaline phosphatase, albumin, lactate dehydrogenase, cholesterol, and bicarbonate levels. With the exception of serum urea, creatinine, sodium, and potassium levels, no significant difference was found between survivors and non-survivors at admission

Table 1 Fournier's gangrene severity index[9]

Variables	High abnormal values					0	Low abnormal values			
	+4	+3	+2	+1			+1	+2	+3	+4
Temperature °C	>41	39–40.9	-		38.5–35.9	36–38.4	34–35.9	32–33.9	30–31.9	<29.9
Heart rate	>180	140–179	110–139		-	70–109	-	55–69	40–54	<39
Respiration rate	>50	35–49	-		25–34	12–24	10–11	6–9	-	<5
Serum sodium, mmol/l	>180	160–179	155–159		150–154	130–149	-	120–129	111–119	<110
Serum potassium, mmol/l	>7	6–6.9	-		5.5–5.9	3.5–5.4	3–3.4	2.5–2.9	-	<2.5
Serum creatinine, mg/100 ml, ×2 for acute renal failure	>3.5	2–3.4	1.5–1.9		-	0.6–1.4	-	<0.6	-	-
Hematocrit, %	>60	-	50–59.9		46–49.9	30–45.9	-	20–29.9	-	<20
White blood cell count, $\times 10^9$/l	>40	-	20–39.9		15–19.9	3–14.9	-	1–2.9	-	<1
Serum bicarbonate, venous, mmol/l	>52	41–51.9	-		32–40.9	22–31.9	-	18–21.9	15–17.9	<15

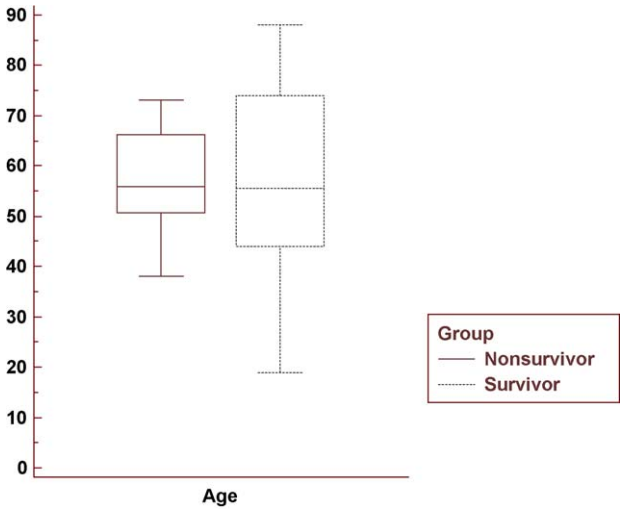

Figure 1 Relationship between age and mortality.

($p = 0.002$, $p = 0.003$, $p = 0.001$, $p = 0.001$, respectively). As can be seen in Table 2, the statistically significant parameters for a bad prognosis and death were as follows: serum urea, creatinine, sodium, and potassium levels at admission and at final evaluation.

Predisposing factors found in 25 patients included perianal disease ($n = 15$, 60%), diabetes mellitus (DM; $n = 14$, 56%), congestive heart failure ($n = 4$, 16%), hypertension ($n = 3$, 12%), chronic renal failure ($n = 1$, 4%), and malignancy ($n = 1$, 4%). Predisposing factors could not be identified in the remaining two patients. Etiologic factors for mortality in our patients were sepsis ($n = 2$) and congestive heart failure ($n = 1$); in two patients no specific etiology could be identified.

The mean duration of hospitalization was 31.54 ± 15.15 (range 14–68) days for survivors and 12.8 ± 14 (range 4–37) days for non-survivors.

Various organisms, alone or in combination, were grown from wound or excised tissue material. In our study *Escherichia coli* ($n = 16$, 51.6%) and *Pseudomonas aeruginosa* ($n = 4$, 12.9%) were the most commonly isolated bacteria. The other organisms isolated were *Staphylococcus aureus*, *Enterobacter cloacae*, *Streptococcus pyogenes*, *Providencia rustigianii*, and *Bacteroides* species. Results of bacteriologic culture are presented in Table 3; four patients were infected with multiple species. All blood cultures were negative except for that of one non-survivor patient, from which *E. coli* was yielded. However, we found that outcome was not related to any specific bacterial species.

The mean admission FGSIS was 5.04 ± 2.49 (range 0–9) for survivors and 13.6 ± 4.61 (range 10–20) for non-survivors ($p = 0.001$); however, the mean FGSIS at final evaluation was 1.45 ± 0.47 (range 0–4) for survivors and 12.2 ± 1.3 (range 11–14) for non-survivors ($p < 0.0001$). The healing rate in the survivor group demonstrates that the FGSIS might be a useful method for evaluating therapeutic options (Figure 2).

Discussion

Fournier's gangrene, a rarely seen synergistic infection originating from the colorectal and genito-urinary systems,

Table 2 Parameters evaluated at admission and at the final evaluation for survivors and non-survivors

Variable	Survivors (n = 22) (min/max)	Non-survivors (n = 5) (min/max)	p- and U-values
Age (years)	53.95 ± 21.49 (19–88)	57.20 ± 12.94 (38–73)	p = 0.05; U = 24 000
Admission time (days)	6.68 ± 3.73 (2–15)	5.4 ± 3.43 (2–10)	p = 0.395; U = 41 500
Body surface (%)	2.29 ± 1.16 (1–4.5)	3.4 ± 1.08 (2–4.5)	p = 0.084; U = 28 000
Number of debridements	1.59 ± 0.66 (1–3)	1.4 ± 0.54 (1–2)	p = 0.599; U = 47 500
Urea (mg/dl)			
Admission	55.59 ± 49.8 (19–246)	83.6 ± 46.17 (37–158)	p = 0.002; U = 6000
Final	34.95 ± 20.9 (18–115)	72.8 ± 20.68 (44–101)	p = 0.003; U = 7500
Creatinine (mg/dl)			
Admission	0.7 ± 0.35 (0.1–1.52)	2.29 ± 1.92 (0.9–5.62)	p = 0.003; U = 8000
Final	0.8 ± 0.26 (0.4–1.7)	1.48 ± 0.69 (0.9–1.71)	p = 0.003; U = 8500
Sodium (mmol/l)			
Admission	131.4 ± 5.07 (119–142)	126 ± 5.78 (120–135)	p = 0.001; U = 3500
Final	135.95 ± 4.29 (128–144)	132 ± 5.09 (129–141)	p = 0.001; U = 2000
Potassium (mmol/l)			
Admission	4.1 ± 0.2 (3.8–4.4)	3.61 ± 0.1 (3.4–3.9)	p = 0.001; U = 4000
Final	4.08 ± 0.33 (3.6–4.8)	3.16 ± 0.28 (2.8–3.6)	p = 0.001; U = 1000
ALP (U/l)			
Admission	284.81 ± 135.99 (86–550)	278.8 ± 31.05 (247–321)	p = 0.779; U = 50 500
Final	241.86 ± 102.28 (123–550)	253.6 ± 60.26 (157–321)	p = 0.435; U = 42 500
Albumin (g/dl)			
Admission	2.32 ± 0.73 (1–3.9)	1.76 ± 0.63 (1.1–2.4)	p = 0.261; U = 37 000
Final	3.21 ± 0.74 (1.89–4.3)	2.1 ± 0.25 (1.8–2.4)	p = 0.010; U = 14 000
LDH (U/l)			
Admission	407.9 ± 122.99 (219–745)	472.4 ± 158.86 (291–684)	p = 0.454; U = 43 000
Final	326.77 ± 68.74 (256–547)	338 ± 81.57 (264–464)	p = 0.803; U = 51 000
Bicarbonate (mmol/l)			
Admission	21.39 ± 3.44 (15.4–27.1)	18.2 ± 3.97 (14–23.4)	p = 0.111; U = 29 500
Final	24.64 ± 1.81 (21–28)	18.58 ± 2.27 (16–21)	p = 0.001; U = 500
Cholesterol (mg/dl)			
Admission	188.4 ± 27.44 (134–241)	176.2 ± 28.41 (140–210)	p = 0.492; U = 44 000
Final	175.4 ± 24.58 (134–241)	176.4 ± 24.13 (145–201)	p = 0.876; U = 52 500
WBC ($\times 10^9$/l)			
Admission	16.719 ± 51.228 (6.390–26.100)	21.420 ± 12.122 (11.300–41.400)	p = 0.492; U = 44 000
Final	10.718 ± 21.277 (7.800–15.200)	16.560 ± 74.393 (8.700–28.300)	p = 0.061; U = 25 000
Hematocrit (g/dl)			
Admission	34.05 ± 6.84 (22.5–47.1)	30.46 ± 5.75 (22.4–38.2)	p = 0.303; U = 38 500
Final	35.96 ± 4.25 (29–44.5)	27.92 ± 2.26 (24.6–31)	p = 0.001; U = 3000
Platelet count ($\times 10^9$/l)			
Admission	285.27 ± 104.12 (102–470)	225.6 ± 123.75 (65–345)	p = 0.532; U = 45 000
Final	289.54 ± 74.79 (156–410)	246.2 ± 100.93 (101–352)	p = 0.618; U = 47 000
FGSIS			
Admission	5.04 ± 2.49 (0–9)	13.6 ± 4.61 (10–20)	p = 0.001; U = 000
Final	1.45 ± 0.47 (0–4)	12.2 ± 1.3 (11–14)	p = 0.0001; U = 000

ALP, alkaline phosphatase; LDH, lactate dehydrogenase; WBC, white blood cell count; FGSIS, Fournier's gangrene severity index score.

begins in the perineal, perianal and genital regions and extends rapidly.[2,10]

In published studies, the median age of patients with FG has been found to be between 46.5 and 63.5 years.[7,11] In our study, we found it to be 54.5 years, similar to Spirnak et al.[12] in 1984; they also found no statistically significant difference in age between survivors and non-survivors. In contrast, Clayton et al.[13] found that survivors were significantly younger than non-survivors, and this was confirmed by Laor et al.[9]

The clinical presentation of the disease starts with a prodromal period of genital discomfort or pruritis, followed by genital erythema with or without crepitus and swelling of the scrotum, often associated with fever and pain. The

Table 3 Bacterial organisms isolated from wound cultures of patients with Fournier's gangrene

Organisms	Total number	Survivors	Non-survivors
Escherichia coli	16	12	4
Staphylococcus aureus	3		
MRSA	1	0	1
MSSA	2	2	0
Streptococcus pyogenes	2	1	1
Pseudomonas aeruginosa	4	2	2
Enterobacter cloacae	3	3	0
Bacteroides spp	2	1	1
Providencia rustigianii	1	1	0

MRSA, methicillin-resistant *Staphylococcus aureus*; MSSA, methicillin-sensitive *Staphylococcus aureus*.

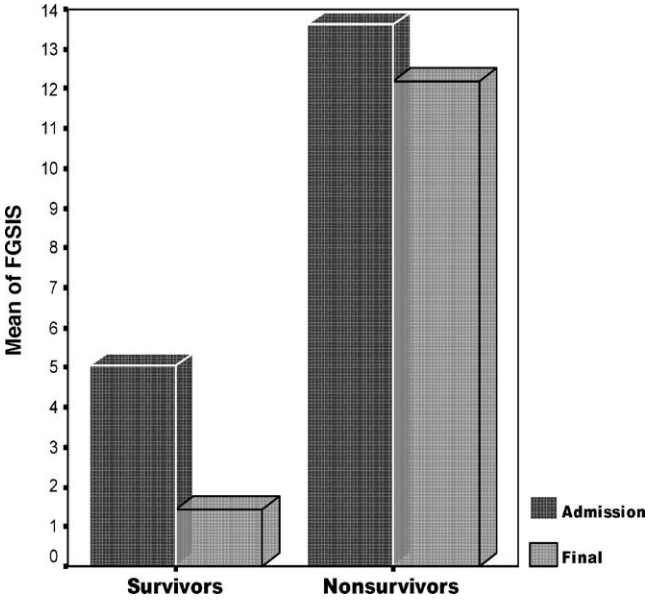

Figure 2 Comparison of Fournier's gangrene severity index score (FGSIS) in survivors and non-survivors.

gangrenous process will lead to drainage of the affected areas and demarcation between viable and dead tissue.[14] In our study most of the patients were admitted with complaints of erythema, perianal or scrotal swelling, pain, and fever. It is important to recognize that patients in the early stages can present with minimal cutaneous manifestations of the underlying infection, making prompt diagnosis difficult. Several symptoms and signs should increase the index of suspicion for a necrotizing subcutaneous infection. For example, an apparent cellulitis that does not respond to appropriate antibiotic therapy should raise a suspicion of FG.[15]

Despite the development of modern intensive care techniques, aggressive debridements, improved wound care, and extensive antibiotic regimens, the reported mortality rates for FG range from 0% to 67%.[2,5,6] FG mortality rates found in Turkey are shown in Table 4.[3,7,10,11,14–16] In the present study, our mortality rate was 18.5%. This high mortality reflects both the aggressive nature of the infection and the destructive effect of accompanying predisposing factors.[7] Severe sepsis, coagulopathy, acute kidney failure, diabetic ketoacidosis, and multiple organ failure are reported as the causes of death in patients with FG. These conditions predispose to septicemia and increasing ischemia by disturbance of the microcirculation, and facilitate advanced circulatory disturbances.[2,11] In our study, DM and perianal disease were the most common predisposing factors, and 54.5% of the survivors and 40% of the non-survivors had DM. Although DM did not affect the outcome in some studies,[9,14] our study indicates that patients with DM are more susceptible to FG.

Surgical management includes the wide incision and drainage of all involved areas and the excision of all necrotic and devitalized skin and subcutaneous tissue.[3,12] Aggressive surgical debridements always suggest a positive effect on survival.[9,12,17] In this study, we found that the number of debridements was not significantly different between survivors and non-survivors. Laor et al.[9] and Palmer et al.[18] also found that repeated surgical debridements did not appear to influence patient outcome. We found that the extent of body surface area involved in the necrotizing process was also not directly related to outcome. This is similar to the findings of Laor et al.[9] and Clayton et al.[13], but not to the findings of others.[11,12,14,19]

Table 4 Comparison of Fournier's gangrene mortality rate in the present study and others from Turkey

Study (Ref.)	Survivors (*n*)	Non-survivors (*n*)	Mortality rate (%)
Uluğ et al., present report	22	5	18.5
Kılıc et al. (3)	19	4	17.4
Tuncel et al. (11)	14	6	30
Ersay et al. (7)	54	16	22.8
Ayan et al. (10)	32	9	21.9
Tahmaz et al. (15)	31	2	6
Korkut et al. (16)	36	9	20
Yeniyol et al. (14)	19	6	24

In our study, when we compared the laboratory values at admission between survivors and non-survivors (Table 2), serum urea, creatinine, sodium, and potassium levels were significantly different. With the exception of these parameters, no other significant differences were observed between the two groups. Serum urea, creatinine, sodium, potassium, albumin, and bicarbonate levels, and hematocrit were significantly different at the final evaluation. Non-survivors had greater serum urea and creatinine levels, and lower sodium, potassium, albumin, and bicarbonate levels and lower hematocrit at the final evaluation. In other studies, such as that of Clayton et al.,[13] only blood urea nitrogen >50 mg/dl was found to be statistically significant for mortality among the parameters studied. Laor et al.[9] reported that patients who survived had significantly greater hematocrit, serum calcium, albumin and cholesterol levels and lower blood urea nitrogen and alkaline phosphatase levels compared with the admission laboratory results. Yeniyol et al.[14] found that lower serum albumin and total protein levels were directly related to mortality. On the other hand Tuncel et al.[11] stated that admission serum biochemical and hematologic parameters do not always reflect severity of the disease. In the light of our results, we found that patients who did not have renal dysfunction at admission were those who survived.

FG begins as an area of infection adjacent to the portal of entry. The infection then progresses to a spreading inflammatory reaction that involves the deep fascial planes. There is a characteristic obliterative endarteritis causing cutaneous and subcutaneous vascular thrombosis and necrosis of tissue. This in turn allows the commensal flora to enter previously sterile areas. Tissue destruction then results from a combination of ischemia and the synergistic action of various bacteria.[1,3,15] FG represents a polymicrobial infection, although not all implicated organisms are necessarily cultured in individual cases. Both aerobes and anaerobes are almost invariably present, but anaerobes are less frequently isolated.[17] Blood cultures are usually negative. Overall, the most commonly isolated species are *Enterobacteriaceae*, especially *E. coli*, followed by streptococcal species; staphylococci, *P. aeruginosa*, peptostreptococci, *Bacteroides spp*, and clostridia are also frequently identified.[9,15] These organisms are present in the normal flora of the gastrointestinal tract and perineum. Paty and Smith[20] reported *E. coli*, Bacteroides and streptococci to be the most common organisms in FG. Laor et al.[9] determined the most common organisms to be *E. coli* and *Streptococcus* species with Staphylococcus and Enterococcus more commonly isolated than Bacteroides. In the present study, similar common bacteriologic agents, such as *E. coli*, Staphylococcus and Bacteroides were isolated in wound cultures from the patients. Chawla et al.[21] stated that if the results of wound culture revealed viridans streptococci, the patients had a longer duration of hospital stay, but this was not significant in our study. Although we isolated few anaerobic organisms, their presence was strongly suspected based on crepitus and subcutaneous emphysema. A comparison of the microorganisms isolated from the patients in our study and from other studies in Turkey is shown in Table 5.[3,7,10,11,15]

The treatment of FG is immediate extensive debridement and a combination of antibiotics, along with treatment of the predisposing conditions. After surgical resection, daily wound care needs to be carried out to fight local infection. A combination of antibiotics targeting all three of the main bacterial groups must be used. Many studies have suggested the use of penicillin against streptococci, metronidazole for anaerobes, and third-generation cephalosporins against staphylococci and *Enterobacteriaceae*.[2,5,6] Aminoglycosides, clindamycin, and chloramphenicol are the antibiotics of choice until the results of culture antibiograms are obtained. If the presence of clostridia is suspected, intravenous penicillin G must be administered.

The FGSI was created by Laor et al.,[9] and in their study the mean FGSIS for survivors was 6.9 and for non-survivors was 13.5. This difference was statistically significant. They found that when a FGSIS of 9 was used as the threshold parameter to predict outcome, those with a score >9 had a 75% probability of death, and an index score of ≤9 was associated with a 78% probability of survival. Chawla et al.[21] reported that the FGSI indicated the likelihood of survival based on variables that could be recorded upon presentation and also provided an efficient way to characterize the severity of presentation and to compare patients. In our study, the mean index score had 100% correlation for survivors and those who died compared with the threshold parameter of 9. This result is confirmed by

Table 5 Comparison of microorganisms isolated from patients in the present study and others from Turkey

Etiological agent	Uluğ et al., present report	Ersay et al.[7]	Ayan et al.[10]	Tahmaz et al.[15]	Tuncel et al.[11]	Kılıc et al.[3]
Escherichia coli	16 (51.6)	28 (17.3)	24 (43.6)	15 (34.9)	4 (26.7)	13 (46.4)
Staphylococcus spp	3 (9.7)	18 (11.1)	15 (27.3)	6 (14.0)	4 (26.7)	8 (28.6)
Pseudomonas aeruginosa	4 (12.9)	17 (10.5)	2 (3.6)	4 (9.3)	-	1 (3.6)
Streptococcus spp	2 (6.4)	26 (16.0)	6 (10.9)	-	-	3 (10.7)
Enterobacter cloacae	3 (9.7)	-	-	-	-	-
Bacteroides spp	2 (6.4)	27 (16.7)	3 (5.5)	-	-	1 (3.6)
Proteus spp	-	13 (8.0)	2 (3.6)	7 (16.3)	-	1 (3.6)
Clostridium spp	-	-	3 (5.5)	-	-	1 (3.6)
Enterococcus spp	-	19 (11.7)	-	1 (2.3)	1 (6.7)	-
Klebsiella spp	-	14 (8.6)	-	6 (14.0)	3 (20)	-
Acinetobacter spp	-	-	-	-	3 (20)	-
Peptostreptococcus	-	-	-	4 (9.3)	-	-
Providencia rustigianii	1 (3.2)	-	-	-	-	-

Results are *n* (%).

other published studies,[7,14,21,22] except the study of Tuncel et al.[11] They stated that the index did not reflect the disease severity and treatment outcome in their patients, however we claim that the FGSI is useful for predicting the outcome of FG.

FG is a rapidly progressive, fulminant infection. Its mortality rate can be diminished by early diagnosis, aggressive surgical intervention, and the use of broad-spectrum antibiotics. In our series, an unstable hemodynamic status at presentation was an important factor that predicted poor outcome. We found that diabetes mellitus and perianal diseases were independent prognostic factors and that renal dysfunction at admission was also an important risk factor for FG. *E. coli* was the most common organism isolated from patient wound cultures. The FGSI is an objective and simple method to quantify the metabolic status and can be used clinically to evaluate therapeutic options and assess results.

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